

Section of Epidemiology and Preventive Medicine

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[October 19, 1956]

The Changing Epidemiology of Sonne Dysentery [Abridged]

PRESIDENT'S ADDRESS

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SONNE dysentery is an epidemiological black sheep. At a time when the intestinal infections as a group have come nicely under control, Sonne dysentery has entered a phase of wild and quite uncontrolled epidemicity. So difficult is it to control that many people would be only too glad to ignore it, if that were possible. Some feel that it is a great mistake to dignify this trifling disease by the name of dysentery, which has such a fearful connotation in the public mind. Many of us at some time or another have turned a blind eye in the hope that we would hear no more about it. Sometimes we have been forced into action for fear of what others may say. The present appears to be a suitable time for taking a look at this disease and studying its recent behaviour.

Although it is probable that the causal organism had been described before, the story of this disease really began with Sonne's description in 1915 of the association of the organism with cases of dysentery in Denmark. The Sonne bacillus was reported in Britain from 1924 onwards, and outbreaks in different parts of Britain from about 1930 onwards. Descriptions of early British outbreaks show many of the features familiar to us to-day. Thus MacGill and Downie in 1932 noted the importance of the school as an epidemic centre, stressing that it was particularly the younger school children who were involved. They thought spread was largely due to close contact, and that mild undetected cases were important in spreading infection. About the same time Pickles (1932) was studying his first outbreak, which took place at the time of the year at which Sonne dysentery epidemics occur nowadays.

However, it was a few years later before things began to warm up. Late in 1937 we read in an editorial in the *British Medical Journal* "During the last few months there has been a notable increase in the incidence of dysentery and diarrhoea, chiefly in children. Small outbreaks have been reported from various parts of England and Wales, but the largest increase has been in London . . ." and later in the same article, "Hitherto in this country Sonne dysentery has for the most part remained localized and sporadic in character, in contrast to its epidemic-endemic prevalence in the U.S.A and Scandinavia". It looks as though in 1937 we were seeing the emergence of Sonne dysentery as a widespread epidemic disease in Britain.

Fig. 1 shows the annual notifications of dysentery in the last twenty-five years. The activity in 1937 and 1938, the beginning of which has just been mentioned, was indeed quite notable by previous standards, although almost insignificant in the light of later events. For those who believe that the common infectious diseases are on the way out this diagram will come as a shock. Not only is dysentery being notified at a very much higher rate than ever before, but there is no indication yet that it has reached peak figures.

How many of the notifications are Sonne dysentery? The Public Health Laboratory Service has published each year a summary of all isolations of *Shigella* by type. In 1945 and 1946, 87% were Sonne. In the next two years incidence was low, and the proportion of Flexner dysentery comparatively high, the percentage of Sonne being 57 and 73. The next year, 1949, the figure was up again to 88%, and since 1950 between 95% and 98% of the isolations have been Sonne. It appears that at least the big changes in notification rate are due to changes in the incidence of the Sonne type.

How much are the higher figures due to improved bacteriological facilities? The growth of the Public Health Laboratory Service, and improving techniques in the isolation of the organism are facts, and it would be strange if they were not reflected in a higher rate of diagnosis of dysentery. The setting up of the service at the beginning of the war may have been responsible for a good deal of the increased incidence between 1940 and 1945. It can have had no relation to the decreased incidence immediately after the end of the war, and it is difficult to believe that any changes in organization and technique that have taken place in recent years can explain the enormous increase in notifications in the 1950s. The increase in the last few years probably represents a real increase in the incidence of Sonne dysentery.

How much of the total incidence of dysentery do the notification figures represent? Some years ago Stocks (1949) estimated that notification of dysentery was only fractional.

This then is the first change in the aspect of Sonne dysentery; an increasing incidence during the last twenty years consisting of successive waves, each of which has receded less than it has advanced, giving the impression of an incoming tide. Whether the tide has yet reached its highest level we do not know. The years 1947-1949 appear to be worth further study as possibly containing the reasons why the wartime epidemic ended and the great prevalence of the 1950s occurred. During this study we shall find that a number of important changes in the epidemiology of the disease did in fact occur just at that time.

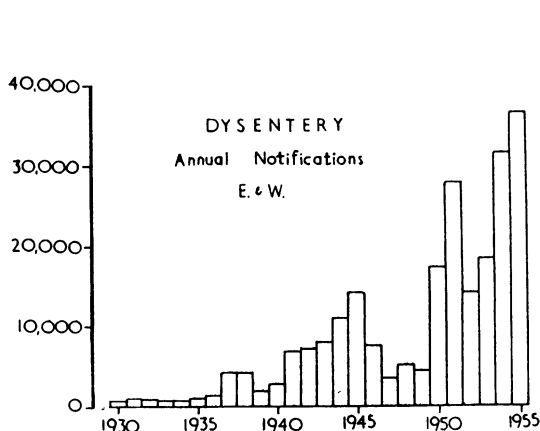
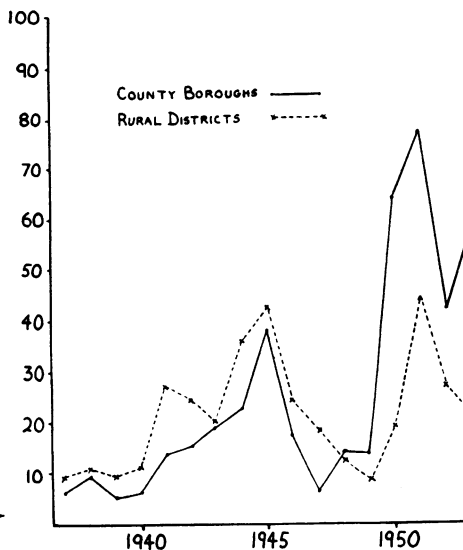


FIG. 1.—Annual notifications in the last 25 years.

FIG. 2.—Dysentery. England and Wales. 1938-54. → Notification rates per 100,000 population.



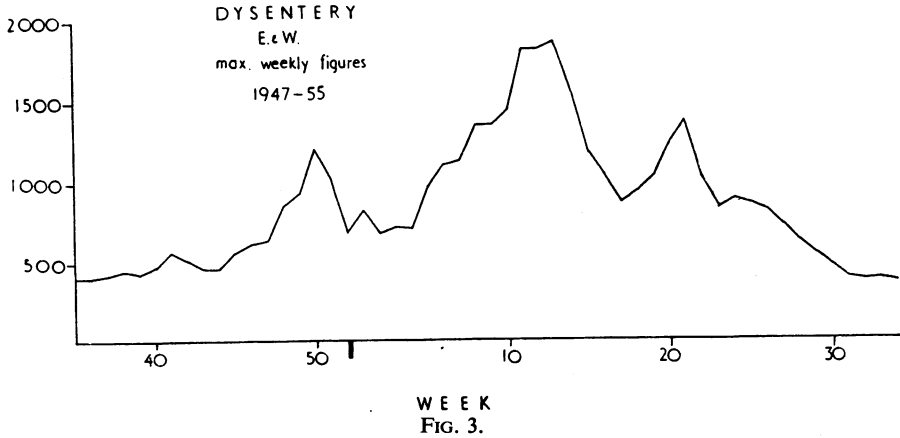
Relation to population density.—During the years in which dysentery has been increasing the experience in the country districts has been quite different from that in the cities. In Fig. 2 the rates for County Boroughs and Rural Districts are shown. It will be seen that up to 1947 the rates in the Rural Districts were higher than those in the County Boroughs, but that thereafter the position was reversed, the disparity between the two types of district being much greater during the last five or six years than before. As far as the Rural Districts are concerned the epidemic of the 1950s has been no greater than the wartime epidemic. It will be noticed that the change-over from rural to urban excess took place during the period of comparatively low incidence after the end of the war. There is no evidence that this change was associated with the wartime evacuation or the return of children to the towns.

The change from rural to urban excess in an infectious disease has been noted before. Greenwood (1935) mentioned it as a change that sometimes happened to a disease when it was in a state of declining incidence, and McFarlan (1951) showed a similar change to be occurring in infective hepatitis in East Anglia in 1946, also during a period of declining incidence. Unlike these examples the change in dysentery occurred during a period of temporary low incidence which was the prelude to the highest incidence ever recorded.

The infectious diseases showing the highest incidence in towns are principally those, such as measles, which spread by personal contact; whereas the most notable example of an infectious disease with a rural excess is enteric fever, a largely food-borne infection. This is a generalization from which there are exceptions. Nevertheless it is suggested that the change from rural to urban excess in Sonne dysentery is consistent with the idea that whereas the disease was at one time spread by a combination of food-borne infection and personal contact, it has now largely abandoned its attempt to be a food infection, and has adopted the far more successful method of personal contact.

Seasonal incidence.—The seasonal incidence of the disease shows equally interesting changes. Fig. 3 gives the maximum notification figure registered in each week of the year from 1947 to 1955. The year's events are shown from nadir to nadir, the diagram running from the 35th week of one year to the 34th week of the next.

In recent years the peak of the dysentery epidemic has been between the 10th and 15th weeks. Following this main peak there is a subsidiary peak about the 20th week. In the curve a peak is also shown about the 50th week, but this is in no way typical and occurred only once during the nine-year period. There is no autumn rise such as is found in enteric fever and food poisoning. This would have been expected between the 36th and the 40th weeks, and such a rise is not seen at all in the dysentery picture from 1947 onwards.



WEEK
FIG. 3.

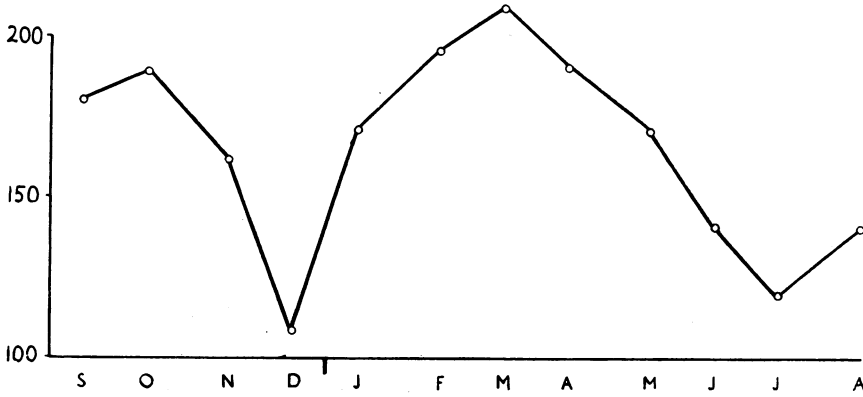


FIG. 4.—Dysentery. England and Wales. Monthly averages of weekly notifications 1941-48. Figures from Glover (1949).

In contrast to this, Fig. 4 is a diagram based on figures published by Glover (1949) for the years 1941 to 1948. Once again the graph begins about the 35th week, but in this diagram monthly averages of weekly notifications are shown. The picture before 1948 was obviously very different from that since that time. The main peak was in March, corresponding to the peak about that time in recent years; but in addition there was, before 1948, a well-marked secondary peak in the autumn, in the months of September and October, at the period of the year in which enteric fever and food poisoning are commonest. The autumn peak, which was present before 1948, has been notably absent in recent years.

This gives further support to the suggestion that the food-borne component has largely disappeared from notified dysentery in recent years. If this is so it is important that it should be generally recognized. Too often do we see the rising figures of dysentery blamed on communal feeding and used unfairly to show how unsatisfactory our food handling methods are.

The idea that dysentery is losing its food-borne component does not rest, however, on indirect evidence only. The figures of reported food outbreaks of dysentery of the Sonne type published by the Ministry of Health and the Public Health Laboratory Service show 41 such outbreaks in the decade 1935 to 1944 compared with only 6 during the period from 1945 to 1954 which was of equal duration but which experienced a very greatly increased incidence of dysentery.

Thus after the year 1947, at which time the incidence of dysentery, particularly Sonne dysentery, was at a low level, the behaviour of the disease has differed in certain respects from that before 1947. It has been more prevalent; it has shown a different relation to population density; it has had a different seasonal incidence; and it has been less associated with food infection than formerly. It is possible that what we have seen is the appearance of a new strain of Sonne dysentery, one that is better adapted to contact infection as a way of life.

Age incidence.—Some years ago Dr. Alison Glover (1947) wrote of dysentery that “the scourge of armies has become the bane of nurseries”, pointing out that the disease was largely one of young children. In recent years there have been a number of reports stressing the way in which the disease spreads quickly and widely in families, and it is obvious that an infection of this type cannot remain exclusively in one age group of the population. Davies (1952) reported the examination of 234 home contacts of cases. Half of them became infected, the proportion varying from 37% of the adults, to 50% of the children aged 8 to 15, and about 80% of those under 7 years of age. These findings have been confirmed by other workers, notably Shaw (1953) and Ross (1955).

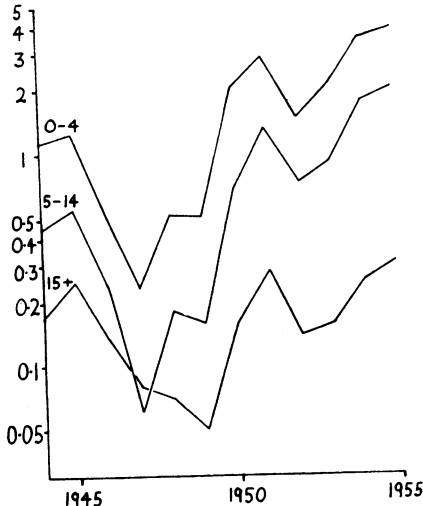


FIG. 5.—Dysentery. England and Wales. Age specific notification rates per 1,000 population.

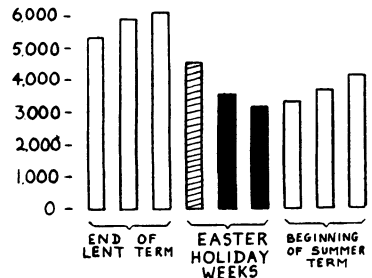


FIG. 6.—Dysentery. England and Wales. Total weekly notifications 1951–55.

It is not surprising, therefore, that a diagram (Fig. 5) of the age specific notification rates over a number of years shows that incidence from year to year has gone roughly parallel in the various age groups. The main exception was in 1947, when for one year the adult rate was higher than that in children of school age. In that year there was a very low incidence of dysentery, coupled with one or two sizable outbreaks of Flexner dysentery in mental hospitals. It is probable that had it not been for the Flexner outbreaks the curve of incidence in the over 15 age group would have shown a dip downwards like those at other ages.

A rise in the percentage of notifications coming in the 5 to 14 years age group in recent years is mainly due to the post-war rise in the birth-rate, but there has also been some degree of closing up between the attack rates in the 0 to 4 years and the 5 to 14 years age groups.

The main focus of activity in Sonne dysentery has now become the primary departments of the schools, where outbreaks are becoming quite frequent.

In view of this association with the schools one would expect to see some evidence of a connexion between the school calendar and the incidence of dysentery. The most constant features of the seasonal curve are the summit of the main peak and the bottom of the trough between that and the following subsidiary peak (Fig. 3). To study the possible relation between these features and the Easter holidays the weekly notifications of dysentery in England and Wales over the five-year period from 1951 to 1955 were taken and arranged not by date, but in relation to the Easter holidays. The dates of the London school holidays were used for this. It is the practice to break up in the middle of a week and to have two and a half weeks' holiday from this time. In Fig. 6 the two black columns are weeks of complete school holiday in London, and the shaded column is half school and half holiday. The height of each column represents the number of weekly notifications in England and Wales.

It is seen that the incidence of dysentery increased steadily during the last three full weeks of the school term; that the beginning of the holiday saw a marked fall in incidence which was maintained through the holiday; and that as soon as the schools reassembled incidence began to rise again towards the secondary peak in the summer term. A somewhat similar picture is seen when figures for individual years are studied. It appears that the main

epidemic is cut short by the break-up of the schools, and that it recovers its momentum when the new term begins. It is not suggested that the schools provide the main impetus for the dysentery epidemic; but there is some evidence that the opening and closing of the schools are responsible for some of the irregularities of the epidemic curve.

Geographical incidence.—When recent incidence in the Registrar-General's Regions is studied we find, in general, a lower incidence in the South and West of the country than elsewhere. This may be due to the tendency already mentioned for incidence to be higher in urban areas than elsewhere.

When comparing one town with another great differences may be due to such causes as variations in the activity of the health department in the search for Sonne, the existence of a public health laboratory in the town, the presence or absence of day nurseries, and many other factors. In most of the large towns there has been a general tendency towards higher incidence in recent years. Nevertheless a high rate in one year has not always meant a high rate for all time. Some of the large cities have managed to get comparatively clear after very high incidence. With regard to the low incidence in the South and West Regions mentioned above it is of interest to note that whereas Plymouth and Portsmouth have both had low rates Cardiff and Bristol have both experienced comparatively high rates. Stoke, Birmingham, and Coventry form a persistent low incidence group in the Midlands which has weathered the storm that has raged about it. It is difficult to find any general rule governing incidence in the large towns in recent years.

The distribution of dysentery in London in recent years has been interesting. Three two-year periods of high incidence were chosen, viz. 1944-45, 1950-51, and 1953-54, and a list was made of the Metropolitan Boroughs in London which had an incidence of more than 1 case per 1,000 population in all three of these periods. Nine Boroughs were in the list. With the exception of Chelsea all the high incidence Boroughs formed a compact block ranging from St. Pancras, Islington, and Stoke Newington in the north, through Finsbury, Shoreditch, Bethnal Green and Stepney further south, to Bermondsey on the south side of the river. The high figures in Chelsea had been inflated by repeated high incidence in a single establishment and a comparatively small population. The compact area in north London was surprising as the distribution of disease and of the various social and environmental factors in London tends to be extremely patchy. The distribution of high dysentery incidence did, however, appear to have some relation to at least one of the environmental indices, namely the average number of persons per room as shown at the Census. With the exception of Chelsea all the high dysentery Boroughs had overcrowding figures (based on persons per room) above the average for the County.

In an inquiry carried out by the Joint Committee of Medical Officers of the London County Council and the Metropolitan Boroughs in 1953 it was found that 16% of the persons notified as dysentery in a year were living in conditions of more than 2 persons per room, the figure for the whole population at the Census being 5.3%. In the same inquiry a higher secondary attack rate in families was found when crowding was present than elsewhere.

PREVENTION AND CONTROL

There are some who think that the prevention of dysentery lies in the washing of hands. Others feel that the disease is uncontrollable. Between these extremes the majority attempt to control the disease with varying degrees of hope and despair, success and failure.

Most will agree that when the disease is present we should do what we can to prevent its spread. Nevertheless this does not mean that we should go on automatically repeating standard procedures regardless of whether they are successful or not. We should consider all our preventive measures in the light of the circumstances of each outbreak, and use only those that have a reasonable expectation of success.

For the present, prevention must, perforce, be limited to the mitigation of local epidemics. There are three main lines of attack on any infectious disease, namely eradication of the reservoir of infection, prevention of transmission of infection, and specific immunization. The prospect of immunization against Sonne dysentery does not look hopeful at present.

The reservoir of infection consists of infected human beings. A great deal of bacteriological examination of contacts is carried out in institutional, nursery, school, and family outbreaks. In bringing to light infected persons who may be in need of treatment this work is obviously valuable; in the prevention of further spread results are uneven, and the reasons for success or failure are not fully understood. The earlier in the outbreak that one begins the better the expectation of success. However, the bacteriological approach is limited by the technical factors involved. Even at the most favourable estimate one cannot expect a bacteriological result until the day after the specimen is taken, and by this time the epidemiological picture may have changed, because Sonne dysentery spreads with such speed. The routine and repeated bacteriological examination of contacts is one of the measures which should be examined with a view to relating the labour involved to the results achieved.

A possible partial solution which deserves further thought is that suggested by Dr. Harry Hawke in a letter to Dr. Bill Brewer, the 25th letter in the Widdicombe file, describing a school epidemic of the Merrygowhimbles (*Lancet*, 1955). He suggested advising the head teacher merely to watch and pray and to see that nobody with diarrhoea cooked or served food; four weeks later he would wish to examine the faeces of the whole school and "smite those *Shigellæ* which linger". This could not be the pattern for all incidents, but on occasions where the initial bacteriological survey discloses that infection has spread wider than expected, farther bacteriological surveys might well be postponed until a time when they can be undertaken more purposefully.

A good deal of the impetus for bacteriological examination of contacts comes from the belief that symptomless excretors are of more importance than cases in the spread of dysentery. There is in fact a certain amount of evidence that the reverse is the case. In the inquiry carried out in London in 1953 to which reference has already been made, it was found that the secondary attack rate in families was considerably smaller when the primary case was a symptomless excretor than when the first case had diarrhoea. Ross (1955) has produced confirmatory evidence on this point.

Once the case or carrier is brought to light the question of putting him out of circulation arises. This often raises the subject of admission to hospital. Easton (1955) has written a strong condemnation of the policy of sending Sonne dysentery patients to isolation hospital, and the more one examines this subject the less justification there appears for using the hospital in this way. Only a very small proportion of cases really require hospital treatment; and removal to hospital can have little effect on the spread of infection. Moreover there are disadvantages in hospital admission, such as expense to the community, the danger of cross infection, and the psychological upset to the child. Unless the home circumstances are totally unsuitable there can be little justification for removal to hospital except in the rare case that must be admitted for treatment that cannot be given at home.

The other form of attack on the reservoir of infection is the direct attack on the organism. It is true that the trials of sulphonamide prophylaxis in day nurseries were not encouraging, but there may be many reasons for this, and this line of attack is surely worth pursuing farther. What is required is a drug that is cheap and effective in quickly and permanently rendering an infected person negative, and which can be given in general practice to sufferers and their family contacts. The disease with which we are dealing is so mild that the drug used must be quite free from side-effects.

The last method of prevention is control of the transmission of infection. The extreme rapidity with which the disease spreads in a closed or semi-closed community makes one wonder whether infection may sometimes be carried by the indirect air-borne route. The organism has occasionally been recovered from dust in the environment of cases. It is not suggested that this is an important method of spread, but we should, perhaps, keep an open mind on the possibility that it may sometimes operate.

If there were ever any doubts about the principal mode of spread of the disease they have surely been abolished by the painstaking demonstration by Hutchinson (1956) of the manner in which children constantly infect their hands from lavatory seats, and the methods by which the seats become infected. This clear demonstration deserves a thorough follow up; a known risk of this kind cannot just be accepted quietly. However, the difficulties should not be underestimated. It is not only a matter of making people wash their hands. The whole subject of the design, method of use, and management of the lavatory is involved. It is a humiliating fact that after 100 years of main drainage the organization of the prevention of lavatory infection, particularly among groups of children, remains one of our biggest problems.

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